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A CASE OF WERNICKE'S CONDUCTION APHASIA WITH AUTOPSY.*

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Next to Broca the greatest credit for our present knowledge of aphasia is due to Wernicke.[1] In his monograph entitled "Der Aphasische Symptomencomplex," published in 1874, some time before the appearance of Kussmaul's work, he gave the first comprehensive sketch of the forms of aphasia, in which the importance of the auditory center for words was recognized for the first time and its location in the upper temporal convolution demonstrated. He clearly showed, from a consideration of the way in which speech is learned combined with a study of cases, that the utterance and especially the selection of words is normally guided by a memory of their sounds, and that this guidance is by means of association impulses from the auditory center to Broca's center; hence the paraphasia which is so conspicuous an accompaniment of the word deafness in cases of destruction of the auditory center. Taking a necessary step further, he concluded that a simple interruption of the path between the two centers must also prevent the auditory center from exercising its normal control over utterance, and he named the resulting defect conduction aphasia *(Leitungsaphasie)*.

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[1] (Wernicke, "Gesammelte Aufsätze und kritische Referate zur Pathologie des Nervensystems," Berlin, 1893, pp. 25-27.)

The symptoms of conduction aphasia are made clear by comparison. He first states that lesion of the auditory center causes word-deafness; loss of imitative speech; paraphasia, in which correct and incorrect words are mingled; agraphia; paralexia on reading aloud and, in the unlearned, loss of comprehension of written or printed words. He then states that conduction aphasia is precisely like this form in its combination of loss of imitative speech, paraphasia, agraphia, paralexia on reading aloud, and loss of comprehension of print; but differs in the absence of word-deafness. In later years he was convinced that loss of comprehension of print or writing would be a necessary symptom in the learned as well as the unlearned. It was assumed that the association path from the auditory center to the motor-speech center was a direct one, through the insula, and "insular aphasia" is sometimes used as synonymous with conduction aphasia.

As a proof that conduction aphasia has a clinical existence, Wernicke published two cases in his original monograph. The first is that of an apothecary, aged 64, who, without any other disturbance, on waking one morning, found himself unable to read or write and, therefore, traveled to Breslau to see an oculist. Not till three days later did his speech become affected. Then his utterance was quite good until inability to recall a name embarrassed and excited him, when it became very much confused. When missing names were suggested to him they were recognized and repeated. Comprehension of what others said was good. On attempting to read aloud there was complete alexia, except for a few very familiar words and there was an entire loss of comprehension of print. He could not name single letters, but objects and pictures were recognized. Spontaneous writing was absolutely lost, but copying remained. There was right homonymous hemianopia. The diagnosis was of a lesion, beginning in the tuber cinereum, encroaching on the anterior perforated space and causing circulatory disturbances in the insula, thus interfering with communication between the auditory and motor-speech centers. The other case presented similar defects in the use of language, together with a probable right hemianopia, but could not be so well observed, as the patient was demented.

In the light of our present knowledge one can have but little doubt that the first of these cases was one of cortical visual aphasia, due to lesion of the angular gyrus, and, in justice to Wernicke, I hasten to add that he evidently has long since changed his mind as to the significance of both cases. Of the first he says in a footnote to his "Gesammelte Arbeiten," that he would now make a very different localization diagnosis. And in another place he says that, as these two cases were complicated, further observations must be awaited before coming to a decision as to the causes of the symptoms of conduction aphasia. Again he says that, as to the location of the lesions which find their functional expression in conduction aphasia only the scantiest and most uncertain material is at hand.

Of a case published by Lichtheim[2] as certainly a clinical example of conduction aphasia, Wernicke[3] remarks that he cannot so regard it, because comprehension of print remained good. This case may also be objected to because the autopsy showed the whole of the Sylvian fossa to be softened and both the third frontal and first temporal convolutions to be somewhat damaged, so the aphasia may be regarded as of a mixed form due to two incomplete lesions.

Notwithstanding the failure of the cases cited to establish this form of aphasia as a clinical and pathological reality, Wernicke seems to have steadfastly adhered to his original theory, confidently awaiting the evidence that would prove it correct. And, on any theory of aphasia which admits that the auditory and motor-speech centers are separate and connected by an association path, his reasoning must be regarded as sound.

It does not appear probable, however, that this association path is a direct one, through the insula. Three years ago, in a paper read before this Association, I[4] remarked that it is only an assumption that this path passes directly forward—an assumption not justified by a study of lesions of the insula—that it much more probably takes a curved course around the posterior extremity of the fissure of Sylvius and that the *Leitungsaphasie* of Wernicke is more likely to be found as a result of lesion of the supramarginal gyrus than of the insula. Recent anatomical authorities might be cited in favor of this opinion, and a case I have observed gives it clinical and post-mortem support.

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[2] Lichtheim, Deutsches Archiv f. klinische Medizin, 1885, Vol. 36, p. 216.

[3] Wernicke, "Gesammelte Arbeiten," p. 121.

[4] Pershing, "Auditory Aphasia," JOUR. NERV. AND MENT. DIS., Sept., 1897.

T. E. P., a man of robust appearance, aged 45, was brought into the hospital in an unconscious condition, Nov. 19, 1897. The meager history obtained indicated that two days before, while driving, he became unconscious and fell from his wagon into the bed of a small stream, where he lay for about 36 hours before being found and removed to the hospital; also that on two previous occasions he had become unconscious, but had apparently fully recovered.

On examination he was found to be completely unconscious, the right arm and leg rigid and tremulous, the head and eyes showing slightly marked deviation to the right. There was no sign of injury to the head; pulse and heart were normal; respirations 20, somewhat stertorous; temperature 99 4-5; urine normal.. A diagnosis was made of a vascular lesion, presumably syphilitic thrombosis, in the left hemisphere, and inunctions and potassium iodide were ordered.

In the course of a week consciousness had completely returned, but the man's talk was a mere jargon, which he shouted in a loud voice. There was also some word-deafness; he generally failed to do what he was told to do, but responded readily when shown by a gesture. No paralysis could be detected. During the second week in the hospital the word-deafness disappeared, and no sign of a cerebral lesion then remained except jargon paraphasia and certain associated defects. Seven weeks after the stroke his condition was noted as follows: Gait, station and the muscular actions generally are normal. On being told in a low tone, without gesture or accompanying change of expression, to perform certain acts he responds readily and accurately. He evidently understands the inquiries made of the nurse and interne as well as of himself, and is eager to answer. In doing so he uses "yes" and "no" accurately, but on attempting to express himself at any length, although the first few words are often intelligible, and apparently appropriate, he soon passes into a fluent but incomprehensible jargon, in which only here and there can a correct word be heard. Rarely the whole of a conventional sentence will be correct and appropriate, as "Very well, I thank you." In trying to answer he never repeats any of the words of the question. He is entirely unable to name familiar objects.

On being asked to read aloud from a newspaper he says that he cannot, but he willingly tries. His reading is markedly paralexic: a few short words are correctly pronounced and the pronunciation of others bears an obvious resemblance to the correct sounds, but these are so mingled with unintelligible sounds that the whole conveys no meaning to a listener. He has no understanding of what he tries to read. The simplest directions given in writing elicit no response, although in studying them he utters some of the words correctly to himself.

He writes his own name quite legibly, only putting an extra "s" on the end. He is absolutely unable to write anything else, either spontaneously or at dictation, and, while the result of all his attempts is illegible, there often recurs a resemblance to parts of his name.

Acuity of vision is good. There is no hemianopia. He has a keen eye for all that goes on about him and often laughs at another patient whose appearance and actions are comical.

The patient's condition remained substantially the same in every respect until Jan. 23, when he suddenly began to vomit and was found to be unconscious. When I saw him next day there was rigidity of the left arm and leg, which were motionless, although the right limbs were moved in a jerky way as though he were resting uneasily. There was conjugate deviation of the head and eyes to the left, and the left pupil was larger than the right. He was quite unconscious. Pulse 64, weak and soft; heart sounds normal. Temperature in right axilla 97.7°, in the left 97.2°. Respiration of the Cheyne-Stokes character. Nails blue. Unable to swallow. Cardiac and respiratory stimulants caused temporary improvement, but death occurred from intercurrent pneumonia Jan. 29.

Autopsy 24 hours after death. Brain firm and well preserved. A general inspection of the membranes and larger vessels revealed nothing abnormal. Careful dissection showed nothing abnormal in the right cerebral hemisphere, but in the left there was a single spot of softening immediately above the fissure of Sylvius, 6 cm. back of the junction of its vertical and horizontal branches. This spot was in the form of an irregular cone, the base being an oval area at the surface, 1.8 cm. in vertical diameter, and 0.8 cm. wide. The long axis of the cone extended inward 2.5 cm. to about the same depth as the floor of the fissure of Sylvius. The pons and medulla were normal, but the dorsal half of the right cerebellar hemisphere was completely destroyed by softening.

After hardening in formalin a more minute dissection of the left cerebral hemisphere was made, especially in the regions of the angular, first temporal and third frontal convolutions, but no additional lesion was discovered. All the arteries examined appeared normal to the naked eye, except one small one in the floor of the fissure of Sylvius near the spot of softening. At a point where this artery divided into a number of branches its wall was extensively and irregularly thickened, so that the lumen was almost obliterated.

Taking the clinical and post-mortem facts of this case together there can be no doubt that the aphasia was caused by the lesion in the left cerebral hemisphere. Motor aphasia was excluded by the large number of words correctly uttered. Visual aphasia was excluded by the absence of hemianopia and the considerable number of words read aloud, either correctly or incorrectly. The defect was clearly a form of auditory aphasia, but was differentiated from other forms by the rapid disappearance of the word-deafness without any change in the paraphasia, paralexia and agraphia. The lesion was small and well defined, and according to the latest anatomical authorities was so situated as to interrupt communication between the first temporal and third frontal convolutions.

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74 TUMEUR CONGÉNITALE DE LA RÉGION LOMBAIRE (Congenital Tumor of the Lumbar Region). P. Piollet (Nouvelle Iconographie de la Salpêtrière, January and February, 1900, 13th Year, No. 1, p. 71).

A congenital tumor of the lumbar region, which does not belong to the class of lipomata found in this region nor is it a variety of spina bifida. The tumor is flat, pediculated, is suspended from the median line at the lower dorsal region, about the size of a hand and the thickness of a finger, and is congenital. When the child began to walk, the tumor gradually increased in size until its weight became a serious embarrassment to locomotion. A quantity of serous fluid exuded from the skin covering the tumor. The skin over the tumor is pigmented. The tumor was removed by operation; it weighed about six kilos. Pathologic examination showed connective tissue formation, malignant in nature by reason of a mucoid tissue. Malignity purely local, susceptible of local recurrence, but not of generalized growth. It is probably a congenital fibromyxoma of the lumbar region, originating from the posterior vertebral arch. It is similar to a solitary molluscum, but differs from it by reason of its malignant evolution. SCHWAB.